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Abstract: Prolonged high-intensity ventilation is associated with the development of rapid shallow breathing with decreased end-inspiratory volumes of all chest wall compartments. During respiratory muscle endurance training using normocapnic hyperpnoea, tidal volume (V(T)) is normally kept constant. The aim of this study was to investigate possible changes in muscle recruitment during constant-V(T) hyperpnoea, to assess potential mechanisms related to rapid shallow breathing. Ten healthy subjects performed 1h of normocapnic hyperpnoea at 70% of maximal voluntary ventilation. Chest wall volume changes were assessed by optoelectronic plethysmography. End-inspiratory (1.08 ± 0.18 versus 0.96 ± 0.27 l, p=0.017) and end-expiratory volumes (-0.13 ± 0.15 versus -0.31 ± 0.19 l, p=0.007) of the pulmonary ribcage decreased significantly and lung function and respiratory muscle strength were reduced (all $p<0.05$). Since with forced, constant V(T) only the inspiratory rib cage muscles were unable to sustain end-inspiratory volume of their compartment, inspiratory rib cage muscles are the most likely candidate responsible for the development of rapid shallow breathing.

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Compartmental chest wall volume changes during volitional hyperpnoea with constant tidal volume in healthy individuals

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ABSTRACT

Prolonged high-intensity ventilation is associated with the development of rapid shallow breathing with decreased end-inspiratory volumes of all chest wall compartments. During respiratory muscle endurance training using normocapnic hyperpnoea, tidal volume (V_T) is normally kept constant. The aim of this study was to investigate possible changes in muscle recruitment during constant-V_T hyperpnoea, to assess potential mechanisms related to rapid shallow breathing. Ten healthy subjects performed 1 h of normocapnic hyperpnoea at 70% of maximal voluntary ventilation. Chest wall volume changes were assessed by optoelectronic plethysmography. End-inspiratory (1.08±0.18 versus 0.96±0.27 l, $p = 0.017$) and end-expiratory volumes (-0.13±0.15 versus -0.31±0.19 l, $p = 0.007$) of the pulmonary ribcage decreased significantly and lung function and respiratory muscle strength were reduced (all $p < 0.05$). Since with forced, constant V_T only the inspiratory rib cage muscles were unable to sustain end-inspiratory volume of their compartment, inspiratory rib cage muscles are the most likely candidate responsible for the development of rapid shallow breathing.

KEYWORDS

Respiratory muscle recruitment, respiratory muscle fatigue, rapid shallow breathing

1. INTRODUCTION

It is widely accepted that inspiratory rib cage muscles, such as inspiratory intercostals, but also scalene, sternocleidomastoid, and other muscles that insert into and expand the rib cage, take over work from the fatiguing diaphragm during exercise when ventilation increases. This is seen in an over-proportional increase of oesophageal compared to transdiaphragmatic pressure generation (Johnson et al., 1993; Mador et al., 1993). Furthermore, an increased perception of respiratory exertion and the adoption of a rapid and shallow breathing pattern are often observed in the course of exercise (Kearon et al., 1991), both of which have been ascribed, at least in part, to rib cage muscle fatigue (Ward et al., 1988; Verges et al., 2006). Shallow breathing leads, however, to inefficient ventilation, i.e. increased dead space ventilation, which may compromise exercise performance at high intensities.

Respiratory muscle training can reduce or delay the development of respiratory muscle fatigue (Romer et al., 2002; Verges et al., 2007; Verges et al., 2009). This in turn may prevent the development of rapid and shallow breathing as shown in some (Spengler et al., 1999; Volianitis et al., 2001; Amonette and Dupler 2002; Romer et al., 2002; Wylegala et al., 2007; Esposito et al., 2010) but not in other (Kohl et al., 1997; Stuessi et al., 2001; Volianitis et al., 2001; McMahon et al., 2002; Holm et al., 2004; Griffiths and McConnell 2007; Verges et al., 2007; Brown et al., 2010; Ray et al., 2010) studies. To gain further insights into potential mechanisms of maintaining tidal volume after respiratory muscle training, we investigated respiratory muscle recruitment during a single respiratory muscle endurance training session. Normocapnic hyperpnoea, which is usually performed for respiratory muscle endurance training, predominantly induces diaphragm fatigue (McCool et al., 1992). In a recent study of

our laboratory, Renggli et al. (Renggli et al., 2008) showed an increase in the ratio of oesophageal to transdiaphragmatic pressure generation during prolonged normocapnic hyperpnoea suggesting a partial takeover of diaphragmatic work by inspiratory rib cage muscles, similar to what was observed during exercise (Johnson et al., 1993; Mador et al., 1993).

Using a different technique to assess respiratory muscle recruitment, i.e. optoelectronic plethysmography (OEP), recent studies in patients with Duchenne muscular dystrophy showed that with progressive weakness of the diaphragm the relative contribution to tidal volume of the abdominal compartment decreases (Lo Mauro et al., 2010; Romei et al., 2011). Thus, specific respiratory muscle fatigue, i.e. a "transient" weakness for instance of the diaphragm, could also lead to changes in relative compartmental contribution to tidal volume. However, results of another study of our laboratory using OEP (Illi et al., 2011), did not confirm the observation made by Renggli et al. during normocapnic hyperpnoea (Renggli et al., 2008). Instead, rib cage muscles appeared to fatigue similarly to the diaphragm, or even earlier, as indicated by the decrease in the end-inspiratory volumes of the corresponding chest wall compartments (Illi et al., 2011). These recent findings thus challenge the assumption that inspiratory rib cage muscles take over diaphragmatic work during normocapnic hyperpnoea. However, the two studies differed in one important aspect: tidal volume was fixed in one study (Renggli et al., 2008) but not in the other (Illi et al., 2011). Thus, the aim of the present study was to assess chest wall volume changes during one hour of normocapnic hyperpnoea with a fixed breathing pattern similar to the study by Renggli et al. (Renggli et al., 2008) and that used during respiratory muscle endurance training sessions.

Based on these previous studies (Renggli et al., 2008; Illi et al., 2011), two hypotheses were tested: 1) during prolonged volitional hyperpnoea with fixed tidal volume, inspiratory rib cage muscles take over inspiratory work from the fatiguing diaphragm, reflected in a change in the relative contribution of the corresponding chest wall compartments to tidal volume, i.e. an increase in upper rib cage contribution and a decrease in lower rib cage and abdominal contribution; or 2) during prolonged hyperpnoea with fixed tidal volume, inspiratory muscle recruitment does not change and the development of global inspiratory muscle fatigue leads to a decrease in end-inspiratory volumes of all chest wall compartments which is either compensated by a decrease in end-expiratory volumes, i.e. expiratory muscles take over work from the inspiratory muscles, or results in task failure.

2. METHODS

2.1. Subjects

Ten healthy, non-smoking subjects (3 men, 7 women) were recruited. All had previously participated in the study on compartmental chest wall volume changes during normocapnic hyperpnoea with unconstrained breathing pattern (Illi et al., 2011). Subjects' characteristics are given in Table 1. All subjects had normal lung function and respiratory muscle strength. Subjects were asked to refrain from strenuous physical activity for two days prior to test days and from any exercise on test days. Drinking caffeinated beverages on test days prior to testing was prohibited, as was food intake 2 h before testing.

Subjects were provided with detailed information about the testing procedures and then gave their written informed consent to participate. The study was approved by the local ethics committee and performed according to the Declaration of Helsinki.

2.2. Protocol overview

Subjects reported to the laboratory on two different occasions, separated by at least 72 h and scheduled at the same time of day. The first session consisted of lung function and respiratory muscle strength measurements, as well as familiarization of subjects with the breathing device for normocapnic hyperpnoea. In the second test session, lung function (forced spirometry) and respiratory muscle strength were assessed, followed by 5 min of quiet breathing and then 1 h of voluntary normocapnic hyperpnoea with a constant breathing pattern. Immediately after, lung function and respiratory muscle strength measurements were repeated, in the same order as prior to hyperpnoea.

2.3. Lung function and respiratory muscle strength measurements

Lung function (vital capacity (VC), forced vital capacity (FVC), forced expiratory volume in 1 s (FEV_1), peak expiratory flow rate (PEF), forced inspiratory volume in 1 s (FIV_1), peak inspiratory flow rate (PIF), and maximal voluntary ventilation in 12 s (12 s-MVV) was assessed according to standard procedures (Miller et al., 2005) using an ergospirometric device (Quark b^2, Cosmed, Rome, Italy) with a calibrated turbine for volume measurements.

Respiratory muscle strength measurements (maximal inspiratory mouth pressure (MIP) from residual volume, maximal expiratory mouth pressure (MEP) from total lung capacity) were conducted according to the current ATS/ERS statement (Green et al., 2002) using a hand held mouth pressure meter (MicroRPM, MicroMedical, Kent, Great Britain). A minimum of five technically-adequate manoeuvres were performed, selecting the highest of three values within no more than 5% variability.

2.4. Normocapnic hyperpnoea

Normocapnic hyperpnoea was performed at a target ventilation of 70% of subjects' individual 12 s-MVV and at a target tidal volume of 60% of subjects' VC with target breathing frequency calculated accordingly. A self-constructed partial rebreathing device described in detail elsewhere (Illi et al., 2011) was used to ensure normocapnia, and breathing frequency was paced by a metronome. Ventilation and end-tidal CO_2 partial pressure ($P_{ET}CO_2$; assessed by a calibrated infrared absorption gas sensor) were measured continuously (Quark b^2). If the breathing pattern deviated from the target, subjects received verbal instructions to adjust either tidal volume or breathing frequency accordingly. In addition, subjects were asked every

5 min to rate their perceived respiratory exertion on a linear scale ranging from 0 (no exertion) to 10 (maximal exertion).

2.5. Optoelectronic plethysmography

Compartmental chest wall volume changes during quiet breathing and normocapnic hyperpnoea (all conducted in standing) were assessed by OEP (BTS, Milan, Italy), a system described in detail by Cala et al. (Cala et al., 1996). Briefly, 89 infrared reflective markers were placed circumferentially on the chest wall between the clavicles and the anterior superior iliac spine. Six infrared cameras (three in front and three behind the subject) traced the three-dimensional coordinates of these markers at a sampling rate of 60 Hz in order to compute the thoracoabdominal volumes using surface triangulation. Chest wall volume was modelled as the sum of the volumes of the three compartments, i.e. the pulmonary rib cage (RCp), the abdominal rib cage (RCa), and the abdomen (AB). The boundaries between RCp and RCa and between RCa and AB were set at the level of the xiphoid and at the lower costal margin, respectively. Specific mechanical properties, including inserting muscles, pressures, and compliance of a compartment, influence volume changes. Changes in volume of the pulmonary apposed RCp ($\Delta V_{rc,p}$) reflect the action of the inspiratory and expiratory rib cage muscles, changes in volume of the diaphragm apposed RCa ($\Delta V_{rc,a}$) reflect the action of the diaphragm and abdominal muscles, and volume changes of the AB (ΔV_{ab}) also reflect the action of the diaphragm and abdominal muscles.

2.6. Data analysis

OEP data of 1 min of quiet breathing, as well as of the first 3 min (start), the middle 1 min (middle), and the last 3 min (end) of normocapnic hyperpnoea were analyzed. The following

variables were derived from OEP using a customized software program (Diamov, designed and developed at the Politecnico di Milano, Dipartimento di Bioingegneria, Italy): tidal volume, breathing frequency, end-inspiratory and end-expiratory chest wall volumes (all derived from or measured at zero-flow points), as well as compartmental volumes (given relative to average end-expiratory volume of the respective compartment during quiet breathing, which was set to zero), and relative contribution of the different compartments to tidal volume.

Data of quiet breathing and of the three time points during hyperpnoea (start, middle, end) were compared using the non-parametric Friedman test. Where a time effect was found, differences between time points were assessed using the Wilcoxon signed rank test, which was also applied to compare lung function and respiratory muscle strength before versus after normocapnic hyperpnoea. Data are presented as mean ± SD. Statistical significance was accepted if $p < 0.05$. Statistical analysis was performed using IBM SPSS Statistics 19.0 (SPSS, Chicago, IL, USA).

3. RESULTS

3.1. Compartmental chest wall volume changes during normocapnic hyperpnoea

A significant time-effect was found for the relative compartmental contribution. As shown in Figure 1, the relative contribution of the abdominal compartment to tidal volume decreased significantly from middle to end ($p = 0.009$) while the contribution of RCp increased ($p = 0.017$). End-inspiratory and end-expiratory compartmental volumes decreased significantly in RCp (Figure 2). The main end-inspiratory and end-expiratory decreases occurred within the first half of the test ($p = 0.013$ and 0.037, respectively). In three subjects, breathing frequency was reduced during normocapnic hyperpnoea (after 9, 25, and 30 min) as they would have reached task failure otherwise. Their end-inspiratory and end-expiratory compartmental volumes were included in the analysis since tidal volume was kept constant.

3.2. Lung function and respiratory muscle strength

In Table 2, lung function and respiratory muscle strength are given before and after normocapnic hyperpnoea. All variables (except FVC) decreased significantly after normocapnic hyperpnoea.

3.3. Respiratory exertion, target ventilation, and end-tidal CO_2 partial pressure

Perceived respiratory exertion increased significantly during normocapnic hyperpnoea (start: 4.0 ± 2.6, middle: 6.1 ± 2.4, end: 7.3 ± 1.8; all $p < 0.01$). Average ventilation (start: 108 ± 17 l·min^{-1}, middle: 106 ± 21 l·min^{-1}, end: 107 ± 21 l·min^{-1}; $p = 0.741$), target tidal volume (start: 2.8 ± 0.6 l, middle: 2.8 ± 0.7 l, end: 2.8 ± 0.7 l; $p = 0.273$) and breathing

frequency (start: 40 ± 5 min^{-1}, middle: 39 ± 6 min^{-1}, end: 39 ± 7 min^{-1}; p = 0.497) remained constant, and $P_{ET}CO_2$ was kept normocapnic (start: 38 ± 3 mmHg, middle: 39 ± 4 mmHg, end: 39 ± 3 mmHg; p = 0.122).

4. DISCUSSION

The present investigation of subjects performing one hour of normocapnic hyperpnoea with a constant breathing pattern suggests that inspiratory rib cage muscles do not take over inspiratory work from the diaphragm. Although the relative contribution of RCp to tidal volume increased, end-inspiratory $V_{rc,p}$ decreased, suggesting additional expiratory adaptations. This compensation for the possibly fatigue-induced decrease in end-inspiratory $V_{rc,p}$ occurred exclusively by increased recruitment of the expiratory rib cage muscles reflected in the decrease in end-expiratory $V_{rc,p}$. Normocapnic hyperpnoea might therefore serve as a training stimulus not only for the diaphragm but also for rib cage muscles.

4.1. Respiratory muscle recruitment during sustained normocapnic hyperpnoea

Subjects in the present study were required to maintain a constantly high ventilation (70% MVV) and tidal volume (60% VC) throughout one hour. This should be kept in mind when comparing the present results with findings in the literature. For example, with increasing exercise intensity or CO_2-induced hyperpnoea and concomitantly increasing ventilation, a progressive increase in end-inspiratory volumes of rib cage compartments and a decrease in the end-expiratory volume of the abdominal compartment is observed (Aliverti et al., 1997; Sanna et al., 1999; Duranti et al., 2004; Romagnoli et al., 2004; Vogiatzis et al., 2005). In the current study, these changes were present right from the start of hyperpnoea where ventilation was already required to be high. In addition, end-expiratory $V_{rc,p}$ was decreased compared to quiet breathing, similar to our previous study where target ventilation was identical (Illi et al., 2011). These initial adaptations – similar to heavy breathing during high-intensity exercise or CO_2-induced hyperpnoea – show that all respiratory muscles, i.e. diaphragm, inspiratory and

expiratory rib cage muscles, and abdominal muscles contribute substantially to the generation of tidal volume during intense volitional hyperpnoea.

The relative contribution of RCp to tidal volume increased in the course of one hour of normocapnic hyperpnoea, while the relative contribution of AB decreased. At first sight, this finding seems to support the hypothesis that inspiratory rib cage muscles take over work from the fatiguing diaphragm. However, the relevance of this change might be questioned as it was not accompanied by significant changes in end-inspiratory and end-expiratory compartmental volumes in the second half of the task and the same average difference in relative contribution to tidal volume did not reach significance in the previous study with changing breathing pattern (Illi et al., 2011). Furthermore, the observed decreases in end-inspiratory and end-expiratory volumes of RCp in the first half of the task suggest that inspiratory rib cage muscles might have fatigued and that maintenance of tidal volume was accomplished by lowering end-expiratory $V_{rc,p}$. Other reasons for the observed changes in end-inspiratory and end-expiratory $V_{rc,p}$ may be strategies to avoid the development of respiratory muscle fatigue and/or to minimise the perception of respiratory effort. However, without fatigue, these strategies would be expected to be present already at the beginning of the task. As lung function variables and respiratory muscle strength were reduced after normocapnic hyperpnoea, the development of inspiratory rib cage muscle fatigue and its compensation on the expiratory side is the most likely explanation to us.

4.2. Mechanical benefit of lowering end-expiratory volume of the pulmonary rib cage

It was hypothesised that end-inspiratory volumes of all compartments might decrease, as observed in the previous study (Illi et al., 2011), resulting in decreases of end-expiratory volumes or task failure. However, in the present study, end-inspiratory $V_{rc,a}$ and V_{ab} were maintained over the entire hour of normocapnic hyperpnoea. The question thus arises why changes in muscle recruitment to maintain tidal volume only occurred on the expiratory side of RCp. Two mechanical benefits might be present for the inspiratory rib-cage muscles when end-expiratory $V_{rc,p}$ is lowered.

i) Although more respiratory muscle work is required to lower the end-expiratory volume of the rib cage than that of the abdomen, since rib cage compliance decreases substantially below functional residual capacity (Grimby et al., 1968) and inspiratory rib cage muscles develop more passive tension than the diaphragm when stretched beyond the optimal length for active force development (Farkas et al., 1985), this work performed during expiration is stored as elastic energy and can be retrieved during inspiration in the form of passive recoil of the rib cage and inspiratory rib cage muscles.

ii) The capacity of the inspiratory muscles to generate pressure is diminished at increased shortening velocities (i.e. flows) and suboptimal length for tension development (i.e. high lung volumes) and might even approach its maximum during intense exercise and/or when respiratory muscle fatigue develops (Leblanc et al., 1988; Johnson et al., 1992). Improvements in the pressure-generating capacity of inspiratory rib cage muscles could thus be achieved by decreasing either shortening velocity or operating lung volume. To avoid task failure in face of the requirement to maintain tidal volume and despite the development of

inspiratory muscle fatigue, subjects needed to lower their operating lung volume. The present data suggest that this lowering of end-expiratory volume took place exclusively in RCp. Since inspiratory rib cage muscles are predominantly responsible for rib cage expanding forces while the diaphragm acts as the main flow generator (Aliverti et al., 1997), it seems intuitive to decrease end-expiratory $V_{rc,p}$ further in order to improve the pressure-generating capacity of the inspiratory rib cage muscles.

4.3 Implications on exercise hyperpnoea

In the present study seven out of ten subjects were able to maintain ventilation at 70% MVV with tidal volume fixed to 60% VC. However, all of these ten subjects spontaneously decreased tidal volume and increased breathing frequency while maintaining the same constant target ventilation during one hour of normocapnic hyperpnoea when breathing pattern was not imposed (Illi et al., 2011). Thus, the question arises, why rapid and shallow breathing developed although most of the subjects would have been able to maintain a constant tidal volume if required to do so. This question is of particular interest for subjects exercising at very high intensities or at altitude where increased dead space ventilation during rapid shallow breathing may further compromise blood oxygen content.

In fact, lowering end-expiratory $V_{rc,p}$ might be even more inefficient than the development of rapid and shallow breathing, which would be supported by the fact that lowering end-expiratory $V_{rc,p}$ below functional residual capacity is associated with respiratory work against a decreased rib cage compliance (Grimby et al., 1968) and increased passive tension in the rib cage muscles (Farkas et al., 1985). Another reason for the development of rapid shallow

breathing might be an increased perception of respiratory effort when tidal volume was required to be held constant. Interestingly, however, perception of respiratory effort was not different between conditions in the present and previous (Illi et al., 2011) study. This suggests that the development of rapid shallow breathing does not minimize the perception of adverse respiratory sensations.

However, the development of respiratory muscle fatigue, in particular of rib cage muscle fatigue (Verges et al., 2006), could have been responsible for the development of rapid shallow breathing in our previous study (Illi et al., 2011). Considering decreases in end-inspiratory $V_{rc,p}$, it seems that inspiratory rib cage muscle fatigue occurred early during the one hour of normocapnic hyperpnoea which would in turn suggest that respiratory muscle training in the form of normocapnic hyperpnoea would not only train the diaphragm but also the other respiratory muscles, in particular the rib cage muscles. This assumption is further supported by the findings of Verges et al. (Verges et al., 2009), who showed a significantly smaller decrease in twitch oesophageal pressure during and after normocapnic hyperpnoea after a period of respiratory muscle endurance training, suggesting reduced development of inspiratory rib cage muscle fatigue. Future studies should thus assess compartmental chest wall volume changes during normocapnic hyperpnoea and high-intensity exercise before and after a period of respiratory muscle endurance training to investigate whether or not this decrease in the development of contractile fatigue of the inspiratory rib cage muscles affects chest wall kinematics and the ability to maintain end-inspiratory $V_{rc,p}$.

4.4. Limitations

Respiratory muscle fatigue was assessed by voluntary manoeuvers rather than pressure measurements during phrenic nerve stimulation. In addition, assessment of the development of oesophageal and transdiaphragmatic pressures over the course of normocapnic hyperpnoea would be helpful to explain the discrepancies between the interpretation of studies calculating the ratio of the pressure-time products and the present results using chest wall volume changes. However, abstaining from invasive pressure measurements in the present study ensured undisturbed respiratory muscle recruitment. Although volitional measures of fatigue may be confounded by the presence of central fatigue, the decrease in end-inspiratory $V_{rc,p}$ with no change in the other two compartments is considered an independent indicator of inspiratory rib cage muscle fatigue. Furthermore, the few studies assessing respiratory muscle fatigue after exercise with both measurement of oesophageal and gastric pressure during nerve stimulation and during voluntary respiratory manoeuvres show a decrease in twitch pressures with smaller decreases (or no significant change) in pressures during voluntary manoeuvres (Johnson et al., 1993; Mador et al., 1993; Babcock et al., 1995; Babcock et al., 1995; Taylor et al., 2006; Kabitz et al., 2008; Taylor and Romer 2008).

Moreover, sample size of the present study was rather low. However, post-hoc power analysis revealed that in most cases, power was close to or above 80% (relative compartmental contributions: RCp 70%, AB 91%; end-inspiratory volume of RCp: 77%; end-expiratory volume of RCp: 80%).

Finally, there might be some concerns about whether or not the three subjects reaching task failure before 1h of hyperpnoea was completed may have influenced the results. For the following reasons, we are confident that this was not the case: i) these three subjects showed changes in relative compartmental contribution that were similar to those of the seven subjects that were able to fulfil the task; ii) end-inspiratory $V_{rc,p}$ decreased in one subject while in two

subjects it did not change; iii) end-expiratory $V_{rc,p}$ decreased in two subjects and increased in one subject.

4.5. Conclusion

In conclusion, during one hour of normocapnic hyperpnoea with a constant breathing pattern, inspiratory rib cage muscles were unable to sustain end-inspiratory $V_{rc,p}$. Expiratory rib cage muscles were increasingly recruited, lowering end-expiratory $V_{rc,p}$, thereby allowing an increased pressure-generating capacity of the inspiratory rib cage muscles. This in turn suggests that inspiratory rib cage muscles play an important role in the development of rapid shallow breathing when subjects are not forced to keep tidal volume constant. If inspiratory rib cage muscle fatigue were the reason for the lowering of end-inspiratory $V_{rc,p}$, a period of respiratory muscle endurance training at a constant and high tidal volume could delay or prevent the development of inspiratory rib cage muscle fatigue and hence the onset of rapid and shallow breathing.

DISCLOSURE STATEMENT

Prof. A. Aliverti is a co-inventor of the Optoelectronic Plethysmography system. Its patent rights are held by his Institution, the Politecnico di Milano, Italy. All other authors have no conflict of interest to disclose.

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FIGURE LEGENDS

Figure 1. Relative compartmental contribution to tidal volume. $\Delta V_{rc,p}$: relative contribution of the pulmonary rib cage to tidal volume; $\Delta V_{rc,a}$: relative contribution of the abdominal rib cage to tidal volume; ΔV_{ab}: relative contribution of the abdomen to tidal volume; TLC: total lung capacity; RV: residual volume; QB: quiet breathing; start: first 3 min; middle: middle 1 min; end: last 3 min of normocapnic hyperpnoea. $^{\#}$ $p < 0.05$ compared to start; * $p < 0.05$ compared to middle.

Figure 2. Individual (grey lines) and mean (thick line) end-inspiratory and end-expiratory compartmental volumes during quiet breathing and in the course of one hour of normocapnic hyperpnoea. $V_{rc,p}$: pulmonary rib cage volume; $V_{rc,a}$: abdominal rib cage volume; V_{ab}: abdominal volume; end-expiratory volume during quiet breathing was set to 0; QB: quiet breathing start: first 3 min; middle: middle 1 min; end: last 3 min of normocapnic hyperpnoea. * $p < 0.05$; ** $p < 0.01$. For clarity, significances between QB and start are not indicated.

Table 1 – Subjects' characteristics

Anthropometric data	
Age [y]	36.6 ± 3.7
Height [cm]	170.9 ± 8.6
Body mass [kg]	63.4 ± 10.4
Lung function	
FVC [% pred]	116 ± 14
FEV_1 [% pred]	111 ± 11
PEF [% pred]	116 ± 17
FIV_1 [% pred]	120 ± 16
MVV [% pred]	131 ± 18
Respiratory muscle strength	
MIP [% pred]	137 ± 36
MEP [% pred]	138 ± 47

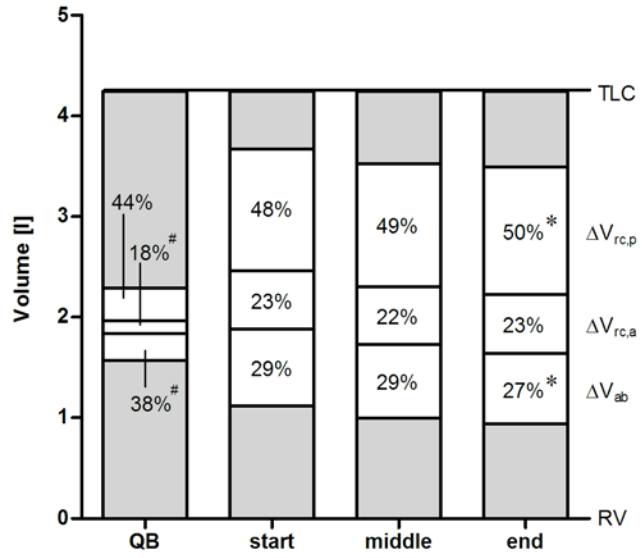
Values are means ± SD. FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 s; PEF, peak expiratory flow rate; FIV_1, forced inspiratory volume in 1 s; MVV, maximal voluntary ventilation; MIP, maximal inspiratory mouth pressure; MEP, maximal expiratory mouth pressure; % pred: % predicted, reference values are from Wilson et al., 1984, and Quanjer et al., 1993.

Table 2 – Absolute values for lung function and respiratory muscle strength measurements pre and post normocapnic hyperpnoea

	pre NH	post NH
Lung function		
FVC [l]	4.8 ± 0.9	4.7 ± 1.0
FEV_1 [l]	3.9 ± 0.6	3.8 ± 0.7*
PEF [$l \cdot s^{-1}$]	9.2 ± 1.8	8.5 ± 1.8*
FIV_1 [l]	4.2 ± 0.7	4.0 ± 0.8*
PIF [$l \cdot s^{-1}$]	6.8 ± 1.5	6.3 ± 1.6*
MVV [$l \cdot min^{-1}$]	164.0 ± 25.7	na
Respiratory muscle strength		
MIP [cmH_2O]	113 ± 21	102 ± 14 *
MEP [cmH_2O]	151 ± 51	128 ± 41 **

Values are means ± SD. NH, normocapnic hyperpnoea; FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 s; PEF, peak expiratory flow rate; FIV_1, forced inspiratory volume in 1 s; PIF, peak inspiratory flow rate; MVV, maximal voluntary ventilation; MIP, maximal inspiratory mouth pressure; MEP, maximal expiratory mouth pressure; na, not available. Significances are indicated for differences between absolute values pre versus post NH. * $p < 0.05$, ** $p < 0.01$.

Figure 1


Volume [l]
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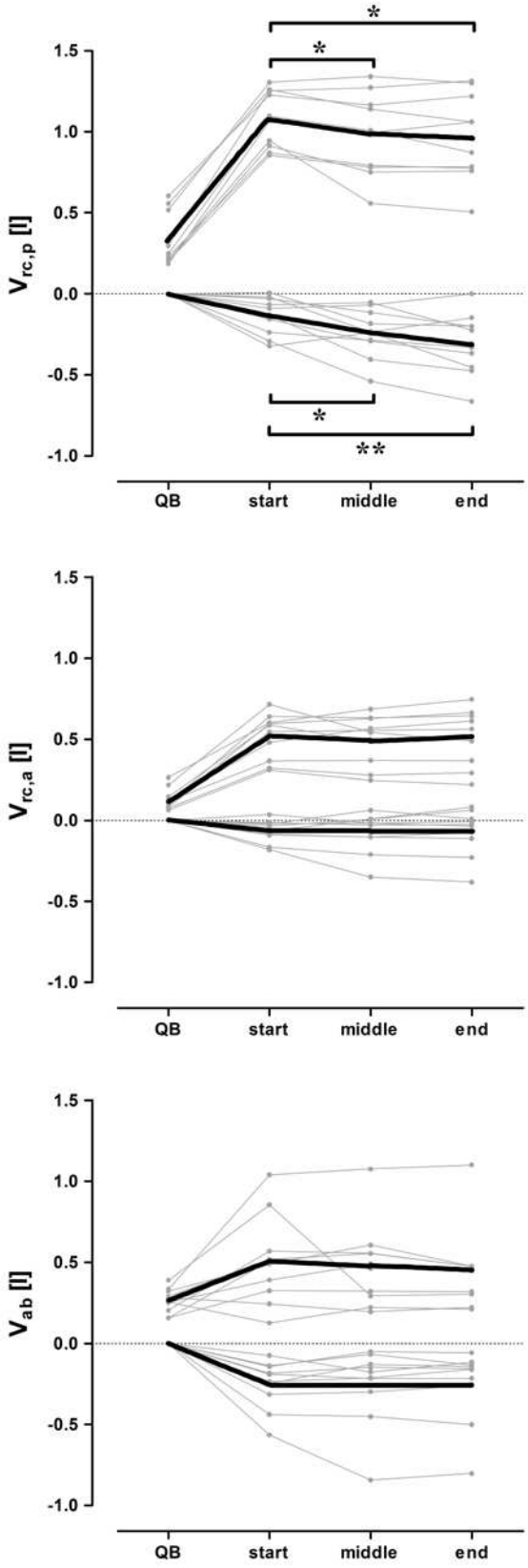
4
3
2
1
0
TLC
RV
44%
18%#
38%#
48%
23%
29%
49%
22%
29%
50%*
23%
27%*
ΔVrc,p
ΔVrc,a
ΔVab
QB
start
middle
end

Figure 2


*
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1.5
1.0
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$V_{rc,p}$ [l]
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$V_{rc,a}$ [l]
QB
start
middle
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1.5
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-1.0
V_{ab} [l]
QB
start
middle
end